Echocardiographic and Phonocardiographic Confirmation of Suspected Caged Mitral Valve Malfunction*

Theodore B. Berndt, M.D.;** Daniel J. Goodman, M.D.;† and Richard L. Popp, M.D.‡

Seven patients studied by echocardiography with and without simultaneous phonocardiography for suspected malfunction of a caged mitral valve prosthesis are presented. In case 1, with inaudible prosthetic clicks, thrombosis of the cage and immobility of the ball were suggested by echocardiographic studies and confirmed at surgery. In case 2, simultaneous echocardiographic and phonocardiographic studies demonstrated wide and variable intervals between the aortic second sound and the opening click and also "sticking" of the ball. In case 3 a thrombus prevented full motion of the ball to the apex of the cage, which was seen on the echocardiogram; while in case 4, with a thrombus within the ventricle and prosthesis, the prosthetic opening click was present intermittently and was associated with only subtle echocardiographic changes. In case 5, echocardiographic studies demonstrated abnormal rocking of the cage secondary to severe prosthetic dehiscence. In case 6, dull prosthetic clicks were to be secondary to a low cardiac-output state. In case 7, with multiple valve prostheses, simultaneous echocardiographic and phonocardiographic studies allowed identification of individual valve sounds and abnormal timing of valve opening. Based on these studies, we believe that echocardiography and simultaneous phonocardiography can yield very useful information in the evaluation of patients with suspected malfunction of a caged mitral valve prosthesis.

Mitral valve replacement with the caged mitral valve prosthesis has provided relief of preoperative symptoms for a large number of patients. Unfortunately, many patients are bothered by postoperative problems associated with valve-related hemolysis, infection, or embolism and with mechanical failure of the prosthesis. Late mechanical failures have been due to ball variance,¹ thrombosis,² periprosthetic leak,³ and left or right ventricular incorporation of the cage.⁴ Characteristic phonocardiographic findings supportive of mechanical malfunction after successful mitral valve replacement have been described.⁵,⁶

We have developed values for a simultaneous echocardiographic and phonocardiographic approach in the evaluation of the normally functioning caged mitral valve prosthesis. In this report, we present five cases of malfunction of a caged-ball mitral prosthesis, one case with multiple caged valve prostheses, and one case with myocardial dysfunction, in order to illustrate the utility of echocardiography with and without simultaneous phonocardiography in the confirmation of suspected valvular malfunction.

MATERIALS AND METHODS

Twenty-one patients (aged 20 to 70 years) in the New York Heart Association's functional classification 1 or 2 and with no evidence of prosthetic malfunction were studied 3 to 48 months (mean, 9 months) after placement of prosthetic mitral valves (Starr-Edwards model 6120). Four of these patients also had aortic or tricuspid caged-ball valve prostheses, or both. The records of these patients were used as the standard of comparison for assessment of records from patients with suspected prosthetic malfunction. Figure 1 demonstrates the measured factors.

Maximal ball excursion (line DE, Fig 1) is measured as the maximal distance between the cage and anterior ball in systole, since these two echoes are not separated during valvular opening in diastole. The intervals between the electrocardiographic Q wave and the closing click, between the aortic component of the second heart sound (A₂) and the opening click, and between the electrocardiographic Q wave and high-velocity closure are measured from the first high-frequency component of the sound or the Q wave, as shown,

CHEST, 70: 2, AUGUST, 1976
to the nearest 5 msec. The sudden rapid movement of the ball away from the transducer after the Q wave of the electrocardiogram is the point of onset of high-velocity closure. The opening velocity and closing velocity of the ball were measured to the nearest centimeter per second in the manner indicated in Figure 1. Values for these measurements were taken as the mean of five cycles in patients with a regular rhythm and of ten cycles in patients with atrial fibrillation.

Echocardiograms were obtained using an ultrasonoscope (Smith-Kline Ekoline 20A) emitting 1,000 pulses per second. The transducer was a 2.25-MHz 0.5-inch active diameter model with an acoustic lens providing beam collimation to 5 cm. Returning signals were recorded on a paper strip chart at 50 and 100 mm/sec, using one of several recorders (Electronics for Medicine DR-8, Honeywell 1856, or Irex 101). Lead 2 of the ECG and a phonocardiogram at the left lower sternal border, using a 120-Hz to 500-Hz band pass filter, were recorded simultaneously with the echocardiogram in most cases. Since it is known that the position of the patient may affect the opening velocity of the ball, all patients were studied in the supine position with the head of the bed elevated to 30°.

Best results are achieved with high damping control settings which produce an abbreviated train of transmitted sound and give apparent increased axial resolution of highly reflective valve structures. An expanded scale is used to enhance visualization and measurement of valve motion once the optimal position for the transducer is found.

The transducer was applied between the lower left sternal border and the left ventricular apex and directed toward the right scapular area. To insure that the sonic beam was parallel with the long axis of the mitral prothesis, the records used in this study were those showing good echoes from the anterior and posterior ball and in which maximal ball excursion is within 1 mm of maximal demonstrated ball excursion in any of the multiple recordings taken from varying transducer positions. Maximal ball excursion is not measured if the sonic beam is moving tangential to the path of the ball, and various values for ball excursion can be obtained, depending on the angle between the sonic beam and the long axis of the prothesis.

Sixteen cases of suspected malfunction of a caged mitral valve prosthesis were assessed by echocardiographic and phonocardiographic examinations during the period of this study. The abnormal findings in these cases were categorized into patterns, and seven representative cases are presented here. The echocardiograms and phonocardiograms were obtained in these patients and analyzed exactly as previously described for the clinically normal patients.

RESULTS

Normally Functioning Values

An average of four transducer positions and sets of recordings (range, three to eight) were made before we were satisfied that maximum ball excursion had been obtained. This procedure required 15 to 45 minutes per examination. In all patients the cessation of the ball's travel recorded by the echocardiogram exactly coincided with the corresponding opening and closing clicks seen on the phonocardiogram (Fig 2). The range of normal values is shown in Table 1. In all cases the mitral cage and sewing ring smoothly moved toward the transducer throughout systole and changed this motion only slightly after the second heart sound. A plateau of cage motion occurred during movement of the poppet. With valvular opening (opening click), the cage began to move smoothly and slowly away from the transducer during diastole. The P wave of the ECG was followed by an abrupt short acceleration of the diastolic cage motion away from the transducer. In those patients with atrial fibrillation, a very small cage motion of this type was recorded as the poppet...
 moved to closure after the QRS wave. Systolic motion of the poppet was exactly like cage motion during the period from the closing click to the second heart sound. Approximately 100 msec after A2, the poppet moved toward the transducer (and cage) to open. The anterior poppet echo apparently contacted the cage’s echo, and fine details of motion were not visible thereafter. Since the posterior poppet echo was spuriously displaced and presented below the sewing ring, the details of the poppet’s motion were assessed from the posterior ball echo; however, the anterior poppet stayed near the apex of the cage in the fully open position throughout diastole in most patients with heart rates above 70 beats per minute. With long diastolic periods the poppet may drift away from the cage apex and toward the echo of the sewing ring. A marked drift of the ball away from the cage’s apex in atrial fibrillation corresponded with a low-intensity closing click (relative to full excursion closure) at the final seating of the poppet in the sewing ring with the onset of systole (Fig 3). If the rhythm is regular and atrial contraction occurs, the poppet will reopen toward the apex of the cage, giving a reopening sound. The poppet usually remains there until after the QRS complex. If there is a prolonged P-R interval, the poppet again may drift away from the cage’s apex until the final high-velocity closure apparently caused by a rise in ventricular pressure. Atrial contraction did not move the poppet to a closed position prior to the QRS.

Table 1—Echocardiographic and Phonocardiographic Data from Patients with Normally Functioning Caged Mitral Valvular Prostheses

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Patients in Regular Rhythm (11*)</th>
<th>Patients in Atrial Fibrillation (10)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (Range)</td>
<td>Mean (Range)</td>
</tr>
<tr>
<td>Opening velocity, cm/sec</td>
<td>25 (17-36)</td>
<td>26 (19-35)</td>
</tr>
<tr>
<td>Closing velocity, cm/sec</td>
<td>51 (26-78)</td>
<td>47 (31-73)</td>
</tr>
<tr>
<td>A2 to prosthetic opening click</td>
<td>100 (70-170)</td>
<td>103 (70-140)</td>
</tr>
<tr>
<td>Electrocardiographic Q wave to</td>
<td>75 (65-100)</td>
<td>78 (50-105)</td>
</tr>
<tr>
<td>prosthetic closing click</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*One patient with junctional rhythm, two patients with first-degree atrioventricular block, and eight patients with normal sinus rhythm.

**In single patient.
complex except when a prolonged P-R interval of above 220 msec allowed postatrial-contraction late-diastolic ball drift.

In our patients with no diastolic ball drift (full excursion closure), the closing velocity and relative intensity of the closing click remained constant, regardless of the prior R-R interval; however, in our patients with diastolic drift, both the closing velocity and the relative intensity of the closing click were inversely related to the prior R-R interval (Fig 3). There was no correlation between the relative intensity of the opening click and factors such as opening velocity, prior R-R interval, or prior interval between A2 and the opening click. Several patients showed multiple opening clicks due to bouncing of the poppet after contact with the cage's apex (Fig 2).

Figure 3. Simultaneous echocardiogram and phonocardiogram from patient with presumed normal mitral prosthetic function and atrial fibrillation, showing diastolic ball drift in first cycle (open arrow) and lack of drift in second cycle with shorter diastolic period. Amplitude of closing click within first heart sound (1) is reduced in first cycle compared with second cycle. Arrowheads bracket amplitude of first heart sound in each cycle. OC, Opening click; AB, anterior ball surface; SR, sewing ring; and PB, posterior ball surface.

Figure 4. Echocardiogram from patient with thrombosed poppet (case 1). Anterior ball (AB) echo is not seen, and cage echoes show same wave form as sewing ring (SR) and posterior ball (PB) echoes. Pattern of posterior ball echoes separated from upper two parallel echoes serves to identify this posterior echo as posterior ball surface. Normal active motion of left ventricular posterior wall and interventricular septal echoes were seen in other portions of record.
Table 2—Summary of Data from Patients

<table>
<thead>
<tr>
<th>Patient, Age, Sex</th>
<th>Clinical Data</th>
<th>Echocardiogram</th>
<th>Phonocardiogram*</th>
<th>Catheterization**</th>
<th>Surgery/Autopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, 49, M</td>
<td>Loss of valvular clicks after stopping anticoagulant therapy for abdominal surgery 4 yr after mitral replacement (Starr-Edwards 6120); no audible valvular clicks; pulmonary congestion</td>
<td>No ball movement independent of cage movement (Fig 4)</td>
<td>No valvular clicks</td>
<td>Mean mitral gradient, 25 mm Hg (MVA, 0.3 sq cm)</td>
<td>Thrombus encasing valve; frozen valve</td>
</tr>
<tr>
<td>2; 47, F</td>
<td>Gradual onset of dyspnea, fatigue, and peripheral edema 4 yr after mitral valve (Smeloff-Cutter) implanted; Variable A2-OC interval heard with atrial fibrillation</td>
<td>Marked beat-to-beat variation of ball movement relative to cage movement (Fig 5)</td>
<td>Wide beat-to-beat variations in A2-OC interval</td>
<td>Mean mitral gradient, 17 mm Hg (MVA, 0.4 sq cm)</td>
<td>Pannus formation within valve, clot on atrial aspect; sticking poppet</td>
</tr>
<tr>
<td>3, 56, M</td>
<td>Intermittent multiple emboli after progression of extreme fatigue and dyspnea 9 yr after mitral valvular replacement (Starr-Edwards 6120); consistently dull opening click; marked peripheral vasoconstriction</td>
<td>Lack of diastolic contact between anterior ball and cage echoes (Fig 6)</td>
<td>Poor quality due to respiratory noise</td>
<td>Mean mitral gradient, 8 mm Hg (MVA, 0.8 sq cm); left ventricular ejection fraction, 10 percent</td>
<td>Thrombus around apex of cage</td>
</tr>
<tr>
<td>4; 44, F</td>
<td>Multiple episodes of palpitations with tachycardia, dizziness, dyspnea, and headache 3 yr after mitral valvular replacement (Starr-Edwards 6120), while receiving anticoagulant therapy; intermittent loss of prosthetic opening click</td>
<td>Intermittent rounding of opening motion of ball in cycles with alteration or loss of opening click; minor changes in ball opening velocity (Fig 7)</td>
<td>Intermittent valvular opening click variable with position and spontaneously</td>
<td>Mean mitral gradient, 15 mm Hg (MVA, 0.7 sq cm); angiogram showed 2 × 2-cm filling defect in ventricle posterior and inferior to cage</td>
<td>Clot in left ventricle adherent to apex of valvular cage; clot extended from sewing ring along posterior ventricular wall</td>
</tr>
<tr>
<td>5, 63, M</td>
<td>Severe dyspnea 1 yr after valve (Starr-Edwards 6120) implanted; edema and pulmonary congestion; apical pansystolic and diastolic rumble audible with dull opening click</td>
<td>Abrupt cage movement with valvular opening and closing; independent ball motion recorded (Fig 8)</td>
<td>High-intensity pansystolic apical murmur; low-frequency diastolic murmur at apex; normal valvular clicks</td>
<td>Excessive cage motion by fluoroscopy; severe peri-prosthetic valvular leak by angiogram</td>
<td>60-percent circumferential dehiscence of valvular sewing ring</td>
</tr>
<tr>
<td>6, 68, M</td>
<td>Severe dyspnea and peripheral edema 1 yr after mitral valvular (Starr-Edwards 6120) and aortic valvular (1200) replacement; dull prosthetic sounds; no murmur</td>
<td>Normal ball and cage motion; marked ball bounce</td>
<td>Prosthetic clicks present</td>
<td>No mitral gradient; cardiac index 1.1 L/min/sq m; LVEDP, 21 mm Hg; poor ventricular contraction</td>
<td>Normal valves at autopsy</td>
</tr>
<tr>
<td>7, 57, F</td>
<td>Marked dyspnea and peripheral edema 7 yr after mitral, aortic, and tricuspid valvular replacement (Starr-Edwards); normal valvular clicks heard</td>
<td>Early mitral ball opening relative to A2 or tricuspid ball motion (Fig 9)</td>
<td>Mitral opening click (identified with echoes) very close to A2 (26 msec)</td>
<td>Left atrial pressures not obtained</td>
<td>Refused by patient</td>
</tr>
</tbody>
</table>

* A2-OC interval, interval between A2 and prosthetic opening click.
** MVA, mitral valve area; and LVEDP, left ventricular end-diastolic pressure.

Suspected Valve Malfunction

Poppet Immobilization. It is difficult to recognize immobilization of the ball (Fig 4 and Table 2) immediately after beginning the echocardiographic examination, since the usual movement of the poppet is not present; however, after prolonged attempts by experienced personnel, the synchronous motion of three sets of echoes suggested a lack of ball motion in the absence of recordable prosthetic valve sounds. The cage and sewing ring basically

CHEST, 70: 2, AUGUST, 1976

CONFIRMATION OF SUSPECTED CAGED MITRAL VALVE MALFUNCTION 225
moved in a normal way. A third set of echoes moving like the cage and sewing ring was recorded in the position usually occupied by the posterior ball echo. No independent movement of the poppet or anterior ball echo was seen. The lack of demonstration of poppet motion assumed significance when the phonocardiogram confirmed the absence of prosthetic sounds and when the posterior poppet echo was recorded moving only with the cage. This is demonstrated in case 1 (Fig 4).

**Poppet Sticking.** The echocardiogram and phonocardiogram together (Fig 5) allowed recognition of the variable interval between A2 and the opening click characteristic of this condition. The maximum beat-to-beat variation in the interval between A2 and the opening click was 20 msec for patients with regular (sinus) rhythm and 30 msec for patients with atrial fibrillation. Beat-to-beat variation beyond this range suggested an impediment to free movement of the poppet.

The example shown in Figure 5 (case 2) had a maximum variation of 130 msec between beats and had many cycles with a value for the interval between A2 and the opening click beyond the 170 msec recorded as the upper limit in the normal group. In this patient, no cycle had the short interval between A2 and the opening click that one would expect with elevated left atrial pressure. The poppet should move to the cage's apex just after the plateau of cage motion is seen. In our cases the movement was variably tardy, as judged by this plateau of cage motion. The consequent pattern of poppet movement looked like a variable shoulder on the opening motion.

**Thrombus within the Valve.** Lack of the normal apparent contact of the anterior ball and cage echoes with valve opening may be due to material preventing full excursion of the ball. The diastolic separation was obvious in case 3 (Fig 6), but motion of the ball relative to the cage otherwise was within normal limits. No phonocardiogram was available from this patient. The clinical impression of muffled valve sounds was clouded by knowing that the patient had severe myocardial dysfunction. This is the only echocardiogram of this type seen during the period of this study.

**Thrombus against the Valve.** Alteration in opening-click intensity and intermittent loss of the opening click with only subtle alterations in ball motion between cycles was present in case 4, with a thrombus within the left ventricle encroaching upon the valve cage. The intermittency of the opening click was recordable and audible at various times during repeated examinations and with altered posi-
tion during the same examination. The echocardiogram showed only a minor change in opening velocity and showed minimal “rounding” of the usual abrupt end of the opening motion of the ball in the cycles with alteration or loss of audible click.

The abnormal opening velocity recorded in this case was present only intermittently. This was the only quantitative abnormality of opening or closing velocity seen in any of the cases.

Valvular Dehiscence. Valve disruption sufficient to cause cage pivoting gives a recognizable pattern of abnormal cage motion. The usual smooth plateau of the cage and ring echoes with ball movement is replaced by an abrupt step or notch. Independent motion of the poppet occurs, but the superimposed excessive cage motion may distort the usual pattern of poppet opening or closing, or both. If the cage pivots slightly, only a notch is recorded on the cage echo. If the valve mobility is great, the whole cage may move in and out of the stationary sonic beam directed in the usual way. The strong cage and ring echoes are recorded easily from many loci on the chest near the left sternal edge and apex in all other patients in whom the valve is normally inserted. Sharp notches in the pattern or total inability to track valvular motion by experienced personnel are rare in the absence of a murmur of mitral regurgitation. Case 5 (Fig 8) had a prominent murmur, and the cage described a maximum arc of 40° on fluoroscopic examination and had a 60 percent circumferential dehiscence of the sewing ring at surgery.

Severe Myocardial Dysfunction. In the presence of pulmonary noises, it may be difficult to differentiate extreme muffling of valve sounds from the absence of clicks by auscultation. Echocardiographic studies aid in this situation. Demonstration of full-excursion poppet movement in such patients may indicate a nonvalvular cause for sound muffling. This is seen in severe impairment of myocardial function with a very low rate of ventricular pressure rise and very low cardiac output. The echocardiographic pattern is notable for marked low-frequency bouncing of the ball, giving the appearance of the
ball floating within the cage. In such cases the opening and closing velocities may be within the range found in the normally functioning hearts we studied. Ball bounce occurs in normal hearts as well, but marked and low-frequency bouncing is exaggerated in low output states. Standard echocardiographic studies of the left ventricle from the parasternal transducer position adds further evidence of poor wall motion in these cases.

**Masked Valve Sound Location.** Patients with both mitral and tricuspid valve replacement may have both valves open synchronously with a single opening click on the phonocardiogram and auscultation. If one valve malfunctions, it may cease contributing to the valvular sound cadence without apparent auscultatory abnormality. An abnormal opening-click time of either atrioventricular prosthesis may be identified by simultaneous echocardiographic and phonocardiographic studies (Fig 9). Absence of the opening click or opening, or both, of either valve may be suspected by this technique.

**DISCUSSION**

There are several reported series of phonocardiographic findings in prosthetic valves.\(^6\)\(^-\)\(^13\) Winters and associates\(^14\) initially described the characteristic echocardiographic pattern of the caged mitral prosthesis in *vivo*. Subsequently Johnson et al\(^15\) established the origin of the various echoes obtained from the Starr-Edwards mitral valve in an *in vitro* study. Others have used simultaneous echocardiograms and phonocardiograms to study the function of such valves.\(^7\)\(^-\)\(^15\)\(^16\) The results in patients with normal valve function in the present series are in good agreement with previous studies with respect to the absolute values for the intervals between A2 and the opening click and between the Q wave and the closing click, and for the opening and closing velocities;\(^15\)\(^16\) however, the maximal beat-to-beat variation of these two specific intervals has received very little comment in prior studies. Maximal normal variation of these intervals in a single patient is important, since variations in the interval between A2 and the opening click have been described in mechanically malfunctioning mitral prostheses.\(^6\)\(^-\)\(^14\)\(^16\) Such variation is especially difficult to interpret in patients with atrial fibrillation. Our data suggest that beat-to-beat variation in this interval greater than 30 msec in patients with atrial fibrillation or greater than 20 msec in patients with regular sinus rhythm should make one suspect mechanical malfunction, especially a sticking valve.

Records from ten patients with regular atrial activity show that atrial contraction did not actively initiate valve closure. With a normal P-R interval, separation of the ball from the cage did not occur until after the onset of the QRS complex. With a prolonged P-R interval, there may be time for some diastolic ball drift toward the closed position after atrial reopening of the valve but before the onset of the QRS complex. This is in agreement with the studies of Siggers et al\(^7\) and some of the patients studied by Hamby et al;\(^17\) however, Hamby and associates\(^17\) suggested that atrial contraction often actively initiated valvular closure in such valves, even with a normal P-R interval.

Patients with atrial fibrillation often show diastolic ball drift towards the closed position, especially during long R-R intervals. Possible mechanisms contributing to this diastolic ball drift towards the sew-
Figure 9. Echocardiogram and phonocardiogram from patient with aortic, mitral, and tricuspid prostheses in place (case 7). Tricuspid valvular motion in upper panel indicates tricuspid opening click (TOC) at distance from aortic closure (ACC) and mitral opening (MOC) labeled in lower panel. Tricuspid closure (TCC) is near QRS peak, while mitral closure (MCC) is during upstroke of QRS complex. Mitral valve motion indicates opening of valve within second sound complex (ACC, MOC). Very narrow ACC-MOC interval was not recognized at bedside or with phonocardiogram alone. LLSB, Left lower sternal border; CPT, carotid pulse tracing; AB anterior ball; SR, sewing ring; PB, posterior ball; and P2, second pulmonic sound.

ing ring include (1) the effect of gravity in the supine position, (2) the late diastolic loss of the antegrade pressure gradient, (3) reversal of the atrioventricular gradient (especially if aortic regurgitation is present), (4) reflected waves from rapid blood inflow; and (5) ring-vortex formation around the valvular poppet. Our study does not permit exclusion of any of these explanations. The latter two mechanisms seem unlikely because of the late diastolic timing of drift onset and the usual lack of ball drift with active atrial contraction in sinus rhythm. The variable intensity of the closing click in the presence of echocardiographic diastolic ball drift is a normal finding.

We are not proposing echocardiography and phonocardiography as the answer for assessing patients developing or maintaining symptoms after valvular replacement; however, we have few other objective means to confirm or deny the clinical suspicion of prosthetic malfunction in these patients. Obviously, the clinical history and physical findings remain the most important criteria by which to judge the patient. Patterns associated with abnormal valvular function help clarify the clinical suspicion.
The combined approach reported here aids in recognizing the grossly abnormal click timing which may be present in such patients. Also, in patients with muffled valve sounds, the demonstration of full movement of the valvular poppet and of poor ventricular wall motion help broaden the physician’s differential diagnosis to include myocardial dysfunction. These noninvasive techniques have been of help in our patients with immobilized valves due to thrombus formation or due to entrapment of tricuspid valves by myocardial trabeculae carneae as the right ventricular cavity shrinks after surgery. The latter situation may be partially masked clinically by the presence of the mitral prosthetic clicks. We have not found clear abnormalities on the echocardiograms or phonocardiograms of patients with small amounts of clot on the mitral or tricuspid valves. We have seen no abnormalities in aortic valvular prostheses in spite of thrombus or vegetations seen at surgery.

The techniques we use give consistent results in patients with normally functioning prostheses. A special caution is in order when proposing lack of normal visualization of a structure as a criterion suggesting an abnormality. Major valve dehiscence resulting in our inability to record the normal pattern of cage, poppet, and sewing ring echoes has been associated with easily audible murmurs of mitral regurgitation and fluoroscopic valve detachment. Lack of poppet movement has been associated with lack of recordable valve clicks and an echo distant from the sewing ring confirming proper technique to record the posterior ball echo (Fig 1). Minor degrees of valve detachment, paravalvular leaks, and thrombus formation may go undetected by these techniques. It is hoped the patterns described here will clarify the situation in some cases.

References